

1017-119

Cardiac Resynchronization Reverses Left Ventricular Remodeling and Reduces Cytokine Activation in Patients With Dilated Cardiomyopathy and Left Bundle Branch Block

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In patients (pts.) with advanced heart failure (HF), dilated cardiomyopathy (DCM) and left bundle branch block, cardiac resynchronization (CR) and appropriate medical therapy improves patient HF symptoms, quality of life and ability to exercise. The purpose of this study was to evaluate the effect of CRT on cardiac structure, function and neurohormones. METHODS: Pts. with HF (NYHA III/IV), DCM (left ventricular end diastolic dimension > 55 mm), and ventricular conduction abnormalities (QRS duration ≥ 140 ms) were implanted with an atrial-synchronous, bi-ventricular pacing system. Atrial-ventricular delay was optimised by Doppler echo (longest left ventricular filling time and greatest cardiac output). NYHA class, QRS duration, six minute hall walk distance, quality of life score, echocardiogram and plasma neurohormones were evaluated at baseline and following 3 mos. of therapy. RESULTS:

	N=25 pts. (22m, 71 ± 9 yrs.)	Baseline	3 month	p-value
Patient Outcomes				
NYHA		2.68 ± 0.75	2.08 ± 0.83	< 0.001
QRS duration (ms)		178 ± 29	148 ± 19	< 0.001
6-Minute Hall Walk (m)		232 ± 163	317 ± 117	0.009
QoL		38 ± 20	18 ± 16	<0.001
Echocardiography				
LVESVI		121.4 ± 51.1	102.0 ± 52.1	0.032
LVEF (%)		30.1 ± 7.6	37.1 ± 9.6	<0.001
CI		2.03 ± 0.68	2.28 ± 0.56	0.012
IVMD (ms)		41.2 ± 23.7	14.3 ± 10.9	0.001
LV MPI		1.07 ± 0.53	0.79 ± 0.51	0.007
Neurohormones				
IGF-1 (ng/ml)		96.7 ± 38.9	118.5 ± 43.6	0.012
TNF-α (pg/ml)		0.018 ± 0.010	0.017 ± 0.010	0.308
sTNFR1 (ng/ml)		1.26 ± 0.49	1.19 ± 0.57	0.306
sTNFR2 (ng/ml)		6.82 ± 2.65	5.98 ± 2.31	0.011

Correlations were found between soluble TNF-α R2 and QoL ($r^2 = 0.166$, $p=0.048$) and soluble TNF-α R1 and LVEF ($r^2 = 0.273$, $p=0.009$). CONCLUSIONS: CRT improves pt. outcomes and appears to reverse some of the adverse effects of heart failure, related to cardiac structural changes and augmentation of the cytokine systems.

1017-120

Reduction of Norepinephrine Levels With Biventricular Pacing but Recurrence of Arrhythmic Events in Patients With Biventricular-ICD and Cardiomyopathy

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It's known that CHF activates the sympathetic nervous system(SNS) and increases serum norepinephrine(NE)in direct proportion to severity of the disease.Emerging data support the use of biventricular pacing(BP)in severe cardiomyopathy(CMP)with reduction of NE levels in follow-up.However if it's real that BP is a promising modality for CHF with LBBB,it's not demonstrated the relation between deactivation of SNS with BP and arrhythmic events(AE)in idiopathic CMP.The aim of the study was to test if the reduction of NE after BP can produce a reduction of VT or VF in pts with idiopathic CMP.METHODS:we enrolled 43 with CMP,LBBB,normal coronary system,maximal medical therapy,spontaneous sustained VT or VF(8pts underwent to ICD)without beta-blockers.We then randomized 20 pts to BP(Medtronic Insync),15 pts to medical therapy and 8 pts to BP-ICD(Medtronic Insync-ICD).In all pts we performed at baseline and after 12 weeks ejection fraction,NE levels and ICD check at 4 and 12 weeks after implantation.All values were expressed as mean ± standard deviation (SD). CONCLUSIONS:in pts with BP or BP-ICD we observed a significant improvement of EF and a marked reduction of SNS activation but all pts who underwent to BP-ICD have VT or VF in follow-up with correct shock by device;4 pts with BP get to sudden death after 4 months.Then we conclude that BP can decrease the NE levels but the deactivation of SNS can't reduce the rate of arrhythmic events in this kind of population and can't reduce the need of antitachycardia devices.

SD(sudden death);VT(ventricular tachycardia);VF(ventricular fibrillation)

	NE(Baseline)	NE(12 wks)
Group I(28 pts:BP and BP-ICD)	600 ± 328 pg/ml	p<0.05 330 ± 130
GroupII(15pts:medical therapy)	570 ± 302 pg/ml	NS 605 ± 260
	AE(1-3 mth)	AE(4-6 mth)
Group I(20pts:BP)		4 SD (20%)
Group II(8pts:BP-ICD)	6 VT and 2 SD (100%)	
Group III(15pts: medical therapy)		1 SD and 2 VT (20%)

POSTER SESSION

1018 Implantable Cardioverter Defibrillator Therapy: Optimal Utilization

Sunday, March 17, 2002, 9:00 a.m.-11:00 a.m.

Georgia World Congress Center, Hall G

Presentation Hour: 10:00 a.m.-11:00 a.m.

1018-107

Performance of a Supraventricular Tachycardia Discrimination Algorithm by an Automatic External Cardioverter Defibrillator in Response to Induced Tachycardia

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Background: An algorithm with three features (Modulation Domain Function, Waveform Factor Analysis and Signal Variability Analysis) is used as a programmable option in an Automatic External Cardioverter Defibrillator (AECED) (Powerheart) to differentiate Supraventricular Tachycardia (SVT) from ventricular tachycardia or ventricular fibrillation within a programmable SVT rate zone. The AECED will withhold therapy delivery in response to tachycardia that is recognized as SVT within that zone and recommend (advisory mode) or deliver therapy (active mode) for faster rates.

Methods: During routine diagnostic electrophysiology studies of 67 pts [Age: 61 yrs (Range 21-92), LVEF: 41% +/- 17%, Males: 39, Bundle Branch Block (BBB) 7], SVT was induced in 25 pts and the AECED then programmed with a detection rate that would result in this tachycardia being within the SVT zone. The advisory mode of the AECED was used to prevent actual therapy delivery. Bipolar right atrial pacing to mimic sinus tachycardia was also tested in an identical fashion in 55 pts.

Results: A total of 31 clinical SVTs were induced [Atrial Tachycardia 8, Atrial Fibrillation 5, AV Reentry 10 and AV Nodal Reentry 8]. Rate detection was accurate in all episodes (Sensitivity 100 %). There were 25 True Negatives, 3 True Positives (episode rate exceeded the SVT zone), 3 False Positives (2 tachycardias with BBB, 1 inducible AV Nodal Reentry Tachycardia with ST segment elevation). Of the 55 right atrial pacing episodes there were 53 True Negatives and 2 False Positives (1 prolonged pacing PR interval with summation in the preceding QRS and 1 with BBB). (Specificity 94%).

Conclusions: This AECED SVT discrimination algorithm functioned extremely well in response to a wide variety of tachycardias (particularly in the absence of BBB) and would be expected to very rarely result in inappropriate suppression of therapy delivery.

1018-108

The Utility of Dual Chamber Electrogram Recordings for the Diagnosis of Clinical Tachycardias

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Background: The availability of the dual chamber defibrillator has allowed the assessment of device performance of various tachycardia discrimination algorithms. Very little data, however, exist about the clinical interpretation of a tachycardia episode on the clinician level in regard to the utility of an atrial electrogram recording and its impact on the correct diagnosis and confidence of that diagnosis. Methods: Dual chamber defibrillator recordings (n=52) with a clear diagnosis were distributed in a blinded and randomized fashion to 5 electrophysiologists, initially with only the ventricular data (Test V) and later with both the atrial and ventricular data (Test AV). The 52 matched pairs of recordings were analyzed with a McNemar's test to determine overall accuracy of diagnosis. Reviewer confidence in the diagnosis via a 5-point Likert scale was assessed with an analysis of variance. Logistic regression determined the relationship of confidence and diagnostic accuracy. Results: Overall accuracy for the specific diagnosis in Test V was 60.8% and 79.2% in Test AV ($p<0.001$). Accuracy in defining the chamber of origin increased from 75.8% to 89.6% with the atrial data ($p<0.001$). Across all reviewers, confidence was significantly greater when the reviewer was correct. Overall mean confidence was greater in Test AV. The odds of a correct specific diagnosis were linearly related to greater confidence in both Test V and AV. Conclusions: The addition of the atrial electrogram allows for improved accuracy for both the specific tachycardia diagnosis and the chamber of origin. Clinician confidence was also enhanced and correlated with accuracy. Thus, the possibility of improved patient care through improved accuracy should be considered when evaluating a patient for a defibrillator.

1018-109

A Prospective Analysis of Changes Stored Intracardiac Electrogram Morphologies After Implantable Cardioverter Defibrillators Shocks

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Background: It is believed that analysis of stored intracardiac electrograms after implantable cardioverter-defibrillator (ICD) shocks is unreliable because of distortions of the electrogram morphology, though the duration and nature of these distortions is unknown. The objective of this study was to prospectively evaluate the effect of ICD shocks on stored intracardiac electrogram morphology and identify variables that may affect these changes. Methods: Twenty-four patients (70% male, 30% female) age 55±12 years undergoing predischARGE or outpatient defibrillation threshold determination received 4 synchronized ICD shocks of 1, 11, 21, 31 Joules (J) at 3-minute intervals. All intracardiac electrograms were recorded from the shocking coils of a dual-coil ICD lead with inte-

grated bipolar sensing (Guidant, Inc., St. Paul, MN) and were digitally recorded to 1000Hz then decimated to simulate the computation environment of an ICD. For each shock event, correlation waveform analysis was used to compare serial post-shock intracardiac electrogram against a baseline template created from the last 5 intracardiac electrograms before the ICD shock. Time to recovery to 90% of baseline was measured in each patient. Results: In the 24 patients studied, electrograms after 120 ICD shocks were analyzed. Nine patients (39%) had significant changes in electrogram morphology after an ICD shock. Time to recovery was 9.3 ± 14.3 sec after an 11J shock, 38.6 ± 28.7 sec after a 21J shock and 45.5 ± 37.1 sec after a 31J shock. No changes in electrogram morphology were observed after a 1J shock. A direct correlation existed between the delivered shock energy and the time to reversion of the intracardiac electrogram to the baseline template ($r=0.45$, $p=0.01$). Patients receiving angiotensin-converting-enzyme (ACE) inhibitor therapy had less electrogram morphology distortions (16.7%) than those not taking ACE inhibitors (63.6%, $p=0.03$). Conclusions: Delivered shock energy and ACE inhibitor therapy can affect distortions in the stored intracardiac electrogram morphology after ICD shocks. This suggests that physiologic factors play a significant role in the observed changes of stored electrogram recordings.

1018-110 Outcome of a Device-Based Atrial Rhythm Control Strategy in Patients With Chronic Congestive Heart Failure and Diminished Ejection Fraction

David Schwartzman, Debra Housel, Srinivas Murali, Atrial Arrhythmia Center, University of Pittsburgh, Pittsburgh, Pennsylvania.

Background: We report an interim assessment of a "device-based" strategy for atrial rhythm control in patients with drug-refractory AF, chronic CHF and reduced left ventricular EF (<40%). Methods: Each patient underwent implantation of a Medtronic Jewel AF (model 7250 device) system. This device incorporates atrial prevention/termination pacing and shock therapies. The device also provides bradycardia pacing and VT/VF therapies. The cohort consists of 32 patients (30 men; mean age 58 yrs; cause of reduced EF: ischemic [18], idiopathic [10], other [4]) with symptomatic, drug refractory AF, who have been followed for 246 \pm 196 days. Successful AF suppression was defined as AF burden <10% during followup. Results: Table shows mean(SD) values for pre-implant and latest followup (post-implant) in pts with successful AF suppression, which was achieved in 27 pts (84%), necessitating adjunct antiarrhythmic drug therapy in 24 of these pts. There have been 8 deaths (all attributable to pump failure; 7 of 8 patients experienced AF suppression), 1 transplant and 1 ventricular assist device implantation. No device-related proarrhythmia was observed. Device therapy for previously undiagnosed VT/VF has occurred in 3 (9%) patients. Conclusions: during short-term followup, device-based atrial rhythm control in this population was feasible, safe, and associated with stable EF, LA/LV volumes, and NYHA class. Therapies for "new" VT/VF were not uncommon.

	Pre-Implant	Post-Implant
EF (%)	30(10)	34(14)
LV Volume (cc)	152(54)	186(68)
LA Volume (cc)	148(53)	151(43)
NYHA Class	2.1(0.9)	2.1(1.0)

1018-111 Importance of Avoiding Nominal Programming to Prevent Inappropriate Implantable Cardioverter Defibrillator Shocks

Andrea M. Russo, Henry Hsia, David Callans, Christa Schorr, Maureen Nicholas, Dusan Kocovic, Francis E. Marchlinski, University of Pennsylvania Health System, Philadelphia, Pennsylvania.

Background: Prophylactic use of implantable cardioverter defibrillators (ICDs) raises increased awareness about the need to avoid inappropriate shocks for supraventricular arrhythmias (SVAs).

Methods: Ninety-one pts without prior clinical sustained ventricular arrhythmias (VAs) received prophylactic ICDs; 46 pts presented with syncope and a dilated cardiomyopathy and 45 pts had no symptoms with coronary disease and inducible ventricular tachycardia (VT). During a follow-up of 21 \pm 18 months, 21 pts (23%) experienced therapy for VAs and 13 pts (14%) for SVAs. We examined clinical VT and SVA rates.

Results: Mean age was 60 \pm 15 yrs and LVEF 25 \pm 9%. Devices were programmed to a "single zone, shock only" configuration in 29 pts, with 2 zones in 5 pts.

Only 2/21 pts (10%) had a clinical VT rate of < 190 bpm (both with inducible VT). The SVA suggested atrial fibrillation 8 pts, atrial tachycardia 3 pts, and sinus tachycardia 2 pts.

Conclusions: (1) Inappropriate shock therapy occurs in 14% of pts receiving prophylactic ICDs during a short follow-up. (2) Despite availability of VT zones and detection enhancements, devices are frequently programmed to a "shock only" zone with a low rate cut-off. (3) Although there is some overlap between VT and SVA rates, VT rates are rarely <190 bpm in the absence of inducible VT. (4) Avoidance of programming to nominal VF detection rates may prevent inappropriate shock therapy for SVAs. The potential role of programming a second zone with detection enhancements needs to be examined.

*p value < 0.01, comparing clinical VT vs. SVA rates

Rate(bpm)	VF zone	VT zone	Clinical VT rate*	Clinical SVA rate*
N =			21 pts	13 pts
Mean \pm SD	182 \pm 13	161 \pm 10	221 \pm 38	179 \pm 14
Range	160 - 214	150 - 175	188 - 308	160 - 200

POSTER SESSION

1019 Noninvasive Testing: Predicting Arrhythmic Events

Sunday, March 17, 2002, 9:00 a.m.-11:00 a.m.

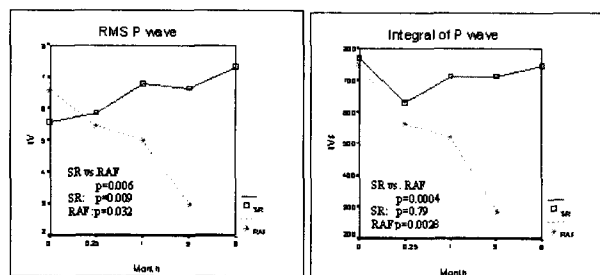
Georgia World Congress Center, Hall G

Presentation Hour: 10:00 a.m.-11:00 a.m.

1019-112 The Evolution of Serial P Wave Signal-Averaged Electrocardiograms Following Direct Current Cardioversion of Atrial Fibrillation: A Prospective Study

Xiao H. Guo, Jan Poloniek, Mark Gallagher, Mohammad S. Hamid, Yi Gang, Marek Malik, A. John Camm, St George's Hospital Medical School, London, United Kingdom.

Background: The evolution of serial P wave signal-averaged ECG (P-SAECG) after DC cardioversion (DCC) of persistent atrial fibrillation (AF) was studied. Method: 60 pts underwent P-SAECG at regular intervals, 5 times over 6 months. Filtered P wave duration (PD), root mean square voltage (RMS) of its terminal 40, 30, 20 ms, entire P wave and integral P wave were obtained using a FFT filter of 40-250 Hz with P wave triggered technique (GE, USA). Results: Of 60 pts (53 men, 66 \pm 10 yrs), 31 (52%), 11 (18%), 2 (3.3%), 2 (3.3%) pts returned to AF (RAF) in 0.25, 1, 3, 6 months respectively. There were no significant differences in age, sex, cardiac disease, AF duration, left atrial size between pts with and without RAF at 6 months. General linear model regression showed RAF group had prolonged PD (157 ± 24 vs 143 ± 17 ms, $p<0.0001$) and lower RMS 40, 30, 20 when compared with pts who maintained sinus rhythm (SR) (5.3 ± 2.0 vs 6.1 ± 3.4 mV, $p=0.007$; 4.3 ± 1.5 vs 5.7 ± 3.2 mV, $p<0.0001$; 3.6 ± 1.4 vs 5.2 ± 3.0 , $p<0.0001$ respectively), these measurements did not change significantly over time in each outcome group. Only RMS-P and integral P evolved against time (Fig). In slope, the RMS-p in SR pts increased ($p=0.009$), whilst a reduction was noted in RAF pts ($p=0.032$) (difference in slopes $p=0.006$). For integral-p, no change in SR group but in RAF slope was significantly decreased ($p=0.0028$), (difference in slopes $p=0.0004$). Conclusion: These differences in evolution suggest that returned AF is preceded by change in serial P-SAECG following DCC.



1019-113 Atrial Fibrillation Recurrence: The Roles Of Hypertension, Duration Of Atrial Fibrillation, and Prolonged Signal-Averaged P Wave Duration

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Background-Prolonged signal-averaged P wave duration (SAPD) has been established as a risk marker for atrial fibrillation (AF). We assessed the risk of hospitalization due to AF recurrence or transition to long-lasting AF in patients with earlier or present AF in relation to the SAPD, clinical characteristics of the patients, and the duration of the AF disease.

Methods-In 111 consecutive patients (71/40 men/women; median age 65 years, range 30-85 years) with earlier or present AF the SAPD was measured at inclusion, and the follow-up time was six months (median 184 days; range 171-437 days). Hospitalization due to AF recurrence or transition to long-lasting AF were regarded as endpoints.

Results-During the follow-up period 33 patients were hospitalized due to AF, and nine patients developed long-lasting AF. History of hypertension, OR=3.67 (95% CI 1.61 to 8.37), duration of the AF disease longer than two years, OR=3.22 (95% CI 1.31 to 7.86), and non-significantly prolonged SAPD above 140 ms, OR=1.87 (95% CI 0.60 to 5.82) were related to an increased risk of hospitalization due to AF relapse or development of long-lasting AF.

Conclusions-Hypertensive heart disease, duration of AF disease longer than 2 years, and prolonged SAPD above 140 ms were risk factors for AF relapse requiring hospitalization or transition to long-lasting AF. The probability of recurrence in patients without risk factors was 9%, with one risk factor 16-27%, with two risk factors 37-54%, and with all three risk factors 74% (Table 1).

Table 1: Probabilities of hospitalisation or transition to long-lasting AF from risk factors (%)

	No hypertension	No hypertension	Hypertension	Hypertension
	< 140 ms	> 140 ms	< 140 ms	> 140 ms
SAPD				
Short duration of AF (< 2 years)	9	16	27	47
Long duration of AF (> 2 years)	24	37	54	74